CORRELATION ANALYSIS OF ELECTROCARDIOGRAMS

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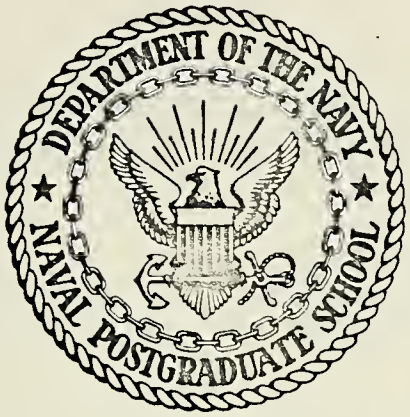

THESIS

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by

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Thesis Advisor: J. A. Miller

March 1972

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Commander, United States Navy
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ABSTRACT

A method for the interpretation of electrocardiographic waveforms by correlation analysis has been investigated. Both the autocorrelation function and the crosscorrelation function are employed in the analysis. The electrocardiographic signals of thirty-six patients, including normal persons and those with specific diagnosed coronary pathology, have been analyzed. Electrocardiographic signs of abnormal heart rate, rhythm, and conduction patterns may be detected by correlation methods; however, the correlated waveform does not appear to be useful in determining the specific abnormality.

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I. INTRODUCTION

Analysis of clinical disorders of the heart beat are of prime interest to the cardiologist. Clinical disorders of the heart beat are either irregularities in the heart's rhythm or abnormalities within the signal components of the heart waveform. These irregularities can be detected by electrocardiograms (ECG) and may be the first evidence of an underlying cardiac abnormality. The ECG is a plot of analog electrical signals from the heart, measured via electrical leads positioned at various locations on the human body.

The ECG is a valuable tool in assisting the physician in his diagnosis of coronary disease. The value of the electrocardiogram arises from the fact that it gives information not obtainable by physical examination, x-ray, or other means available at the present time. The ECG records the electrical stimulus or impulse that arises in each portion of the heart together with the sequence with which it is transmitted to other portions of the heart. Thus in most instances it is possible to determine the source of the rhythm and the frequency with which the impulses arise. When the ventricular musculature is normal and intact, the ECG assumes a characteristic, readily recognizable form. When the musculature is altered, by either pathologic or physiologic factors, the tracing may show changes which will enable recognition of what has occurred.

Contemporary methods of analysis of ECG range from time-consuming visual analysis by the physician to expensive computer-processed pattern recognition techniques. A computer processed ECG is intended

to be an aid to the physician interpreting an ECG for patient diagnosis, care, or clinical study purposes. As such, the computer processed electrocardiogram should be viewed as a screening tool that provides a consistent answer when defined wave patterns are present. This tool is expensive, for example an IBM 1800 data acquisition and control system, which can simultaneously monitor the condition of five patients, rents for approximately $8,000 a month.

Although there are computer systems which are cheaper than the IBM 1800, none currently appears to fit the needs of a small (75 bed) hospital. Therefore there is need for a relatively low cost system which would aide the physician in the analysis of clinical disorders of the heart beat.

Clinical disorders of the heart beat are observed as deviations from normal heart rhythm or deviations from the normal ECG pattern. Since rhythm is a temporal function, and correlation analysis provides a quantitative measure of the degree of similarity between waveforms as they are shifted relative to one another in time, the autocorrelation and crosscorrelation functions appear promising as methods of analysis of ECG signals. The present work reports on an attempt to employ correlation analysis in the interpretation of ECG signals.

II. A BRIEF DESCRIPTION OF THE HEART AND ELECTROCARDIOGRAPHIC ANALYSIS

The heart is an efficient two stage muscular pump. Even during sleep the heart pumps approximately two ounces of blood with each beat, nearly five quarts per minute or about 75 gallons per hour. When an individual is moderately active the heart may double its output, and during strenuous exercise the heart's output may reach 250 gallons per hour.

The heart is shaped like a blunt cone about the size of the fist of the individual. It rests on the diaphragm between the lower part of the two lungs. The apex of the cone points downward, forward, and to the left, about two-thirds of the whole organ being to the left of a plane passing through the vertical median of the individual.

The wall of the heart is composed of three layers, an outer epicardium, a middle myocardium, and an inner endocardium. The epicardium consists of layers of transparent tissues. The myocardium is composed of layers of bundles of cardiac muscle with a minimum of other tissue except for the blood vessels. The endocardium is the interior lining of the heart.

The heart consists of four chambers: two larger ventricles with thick muscular walls making up the bulk of the organ and two smaller atria with thin muscular walls. The septum which separates the ventricles also extends between the atria, subdividing the whole heart into what are called left and right halves or sides of the heart. Figure 1 illustrates the anatomic components of the heart.

As illustrated in Figure 2, the right ventricle is wrapped half-way around the left ventricle. The cause of this is the difference in pressures, a ratio of approximately 6:1, developed by the two ventricles during systole - the period of contraction of the heart muscles during the cardiac cycle. Because the left ventricle contracts with extreme force in comparison with the right ventricle, the left ventricle assumes a spherical shape, and the septum protrudes into the right heart. Each side of the heart pumps essentially the same quantity of blood; therefore, the external wall of the right ventricle bulges far outward and extends around a large portion of the left ventricle, in this way accommodating about the same quantity of blood as the left ventricle.

The period from the end of one heart contraction to the end of the next contraction is called the cardiac cycle. This cycle consists of a period of relaxation called diastole followed by a period of contraction called systole. During the cardiac cycle the right ventricle pumps oxygen-depleted blood from the veins to the lungs via the pulmonary artery. There, blood discharges carbon dioxide and absorbs oxygen. Then, traveling through the pulmonary veins to the left atrium and left ventricle, the blood is pumped via the systemic circulation complex to all parts of the body. The systemic circulation supplies all the tissues of the body except the lungs, with blood flow.

The adult human heart normally contracts at a rhythmic rate of seventy-two beats per minute. Figure 3 (a) illustrates the special excitatory and conductive tissue system of the heart that controls these cardiac contractions. The figure shows: the S-A node in which the normal rhythmic self-excitatory impulse is generated, the A-V node in which the impulse from the atria is delayed before passing into the

ventricles, the A-V bundle, which conducts the impulse from the atria into the ventricles, and the left and right bundle branches.

The sequence of cardiac excitation is illustrated in Figures 3 (b) and (c). To initiate each heart contraction a low intensity electrical impulse is generated in a small strip of specialized muscle on the posterior wall of the right atrium called the sino-atrial (S-A) node. The impulse spreads immediately into the surrounding atrial muscle and is conducted in all directions at a velocity of about 0.3 meter per second. At the same time a few specialized atrial muscle fibers conduct the impulse with extra speed directly from the S-A node to the atrioventricular (A-V) node (the various nodes and bundles which comprise the conduction system of the heart are basically highly specialized muscle fibers). The A-V node is the bulbous end of a bundle of Purkinje fibers - specialized fibers that rapidly conduct the cardiac impulse. The Purkinje fibers, after originating in the A-V node, form the A-V bundle, which then threads between the valves of the heart and thence into the ventricular septum as shown in Figure 3 (a). The A-V bundle divides almost immediately into the left and right bundle branches that lie beneath the endocardium of the respective sides of the heart. The terminal Purkinje fibers in the bundle branches enter the ventricular muscle from the endocardial surface and terminate on the muscle fibers. The cardiac impulse is then transmitted through the ventricular muscle mass by the ventricular muscle fibers at a velocity of 0.3 to 0.4 meter per second.

Electrical currents generated by the cardiac impulses spread from the heart into surrounding tissue and then to the surface of the body. An ECG is a recording of these electrical potentials generated by the

heart and is obtained by placing electrodes at various locations on the body. For the normal twelve electrocardiographic circuits, which are termed leads, there are ten electrode positions. Figure 4 illustrates which electrodes are used to obtain the different lead waveforms.

Figure 4 (a) illustrates electrical connections between the limbs and the electrocardiograph for recording ECG from the three standard limb leads. The leads are connected as follows:

Lead 1: the negative terminal of the electrocardiograph is connected to the right arm and the positive terminal to the left are electrode.

Lead 2: the negative terminal of the electrocardiograph is connected to the right arm electrode and the positive terminal to the left leg.

Lead 3: the negative terminal of the electrocardiograph is connected to the left arm electrode and the positive terminal to the left leg.

The augmented unipolar limb leads - AVR, AVL, and AVF - eliminate the double exposure characteristics of the standard leads and obtain a picture of the electrical activity as seen from a single extremity only. The electrodes are placed as shown in Figure 4 (b). In this type of recording, two of the limbs are connected through electrical resistances to the negative terminal of the electrocardiograph while the third limb is connected to the positive terminal. When the positive terminal is on the right arm, the lead is termed the AVR; when on the left arm, the lead is termed the AVL; and when on the left leg, the lead is termed the AVF.

The three standard leads and the three augmented unipolar limb leads all record the electrical activity of the heart as viewed from its lateral aspects and do not adequately reflect the electrical activity of the anterior and posterior surfaces of the heart. This latter information is obtained by placing the exploring electrode at six different locations on the chest. These six electrode positions result in the six unipolar precordial leads. The exploring electrode is connected to the positive terminal of the electrocardiograph, and the negative electrode, called the indifferent electrode, is connected simultaneously through electrical resistances to the right arm, left arm, and left leg, as shown in Figure 4 (c). The indifferent electrode is unaffected by potentials developed by the heart since the three electrodes are approximately equidistant from each other and from the heart, and therefore tend to cancel each other.

During all recording an additional electrode is applied to the right leg. This electrode does not enter into the electrocradiographic picture but serves as a common ground for man and the machine.

The typical ECG is composed of a P wave, a QRS complex, and a T wave (Figure 5). The P wave is caused by electrical currents generated as the atria depolarize prior to contraction. The QRS complex, actually three separate waves - the Q wave, the R wave, and the S wave - is caused by currents generated when the ventricles depolarize prior to contraction. The T wave is caused by currents generated as the ventricles recover from the state of depolarization.

The distinction between depolarization and repolarization is important since the majority of the nerve and muscle fibers in the heart are excitable - that is, capable of transmitting electrochemical

impulses along their membranes. In the resting state the inside of the nerve or muscle cell is at a negative potential where the outside potential of the cell is positive. During depolarization there is a reversal of potential and the outside wall becomes negative. When the cell is repolarized the cell returns to its normal resting potential. Before contraction of muscle can occur, a depolarization wave must spread through the muscle. Therefore, the P wave, associated with the atria, occurs at the beginning of contraction of the atria, and the QRS wave, associated with the ventricle, occurs at the beginning of the contraction of the ventricles. The atrial repolarization wave is not seen on an ECG since it occurs at the same time the stronger QRS is recorded. However, the ventricular repolarization wave, the T wave is visible.

The five waves described may vary in amplitude and duration and a series of waves may vary in frequency. These parameters - amplitude, duration, and frequency - form the basis for electrocardiographic knowledge about the position and condition of the heart.

III. THE CORRELATION FUNCTION

Correlation analysis is concerned with the determination of the degree of similarity between two signals or between successive samples of a repetitive signal. Correlation analysis leads to the concept of a correlation function which involves time as a parameter. This follows from the notion that the similarity of two signals depends on their being temporally congruent. Two types of correlation functions may be defined corresponding to the problems of comparing a signal with a later repetition of itself, autocorrelation, or comparing a signal with one from an alternate source, crosscorrelation.

The principal requirements for application of correlation techniques are:

1. The signals have to be stationary. That is, a random process is stationary if its finite-dimensional distributions are invariant to arbitrary translations of time. (It is not necessary that a process be stationary for all time but only for some observation interval which is long enough to be suitable for a given problem.)

2. The duration of each signal must be at least one order of magnitude greater than the period of the lowest frequency component of interest.

The autocorrelation function $R_x(\tau)$ is defined by:

$$R_x[\tau] = \lim_{T \to \infty} \frac{1}{2T} \int_{-T}^{T} x(t)\, x(t+\tau)\, dt \qquad (1)$$

where 2T is the time interval of integration.

The autocorrelation function may be interpreted as a point-by-point multiplication of a waveform, x (t), with a later version of itself, in which the delay time, τ, is a parameter, followed by an integration or summation over all time. The following mathematical properties are associated with the autocorrelation function:

1. The autocorrelation funciton is an even function. It is symmetrical about $\tau = 0$, i.e.:

$$R_x(\tau) = R_x(-\tau)$$

2. The value at $\tau = 0$ represents the total signal power, both AC and DC components. This is the mean square value of the signal x(t), and will be a maximum when $\tau = 0$, i.e.:

$$R_x(0) = \text{mean square value } \overline{[x^2(t)]} \geq R_x(\tau)$$

3. The value of $R_x(\tau)$ as $\tau \rightarrow \infty$ approaches the DC power of the signal x(t), i.e.:

$$R_x(\infty) = (\text{average DC component})^2$$

The crosscorrelation function $R_{xy}(\tau)$ is defined by:

$$R_{xy}[\tau] = \lim_{T \rightarrow \infty} \frac{1}{2T} \int_{-T}^{T} x(t)\, y(t+\tau)\, dt \qquad (2)$$

Equation (2) may be interpreted as a point-by-point multiplication of a waveform by a delayed version of a second waveform, in which the delay time is a parameter, followed by an integration or summation over all time. The crosscorrelation function has the following mathematical properties:

1. The crosscorrelation function displays symmetry about the ordinate when x and y are interchanged, i.e.:

$$R_{xy}(-\tau) = R_{yx}(\tau)$$

2. The square of the magnitude of the crosscorrelation function is never greater that the product of the power contained in the two signals, i.e.:

$$\left| R_{xy}(\tau) \right|^2 \leq R_x(0) \cdot R_y(0)$$

3. The magnitude of the crosscorrelation function is never greater than the average of the power contained in the two signals, i.e.:

$$R_{xy}(\tau) \leq \frac{1}{2}\left[R_x(0) + R_y(0)\right]$$

IV. EXPERIMENTAL PROCEDURE

As illustrated in Figure 6, electrocardiographic data from a Hewlett Packard model 1511A or a Hewlett Packard model 1513A electrocardiograph were recorded on a Telex 434 tape recorder. The data were then analyzed by a SAICOR SAI-42, correlation and probability analyzer. The correlated data were plotted on a Variplotter model 1100. To verify the quality of the signal analysed, an oscilloscope was used to monitor the input to the analyzer. The frequency response of the entire system was flat within 3 db in the frequency ban of interest (0.05 to 100 hertz).

The correlation analyzer is an all-digital instrument capable of on-line real-time computation. The digital processing technique employed by the instrument reduced Equations (1) and (2) to the following forms:

$$R_x[\tau] \cong \frac{1}{N} \sum_{K=1}^{N} X_K X_{K+\tau} \tag{3}$$

$$R_{xy}[\tau] \cong \frac{1}{N} \sum_{K=1}^{N} X_K \cdot y_{K+\tau} \tag{4}$$

where numerical integration is limited to the positive half of the time inteval, T, divided into N segments, and τ is the delay.

The sources of error in the digital correlation technique arise primarily from the sampling and quantizing process and the finite intergration time. The accuracies, therefore, are difficult to

analyze since the signal statistics, bandwidth and other parameters must be chosen. For the analysis of the ECG signals N was taken as 2048 and τ was taken at 10 ms.

It can be shown[1] that using random noise with a bandwidth of B, the normalized standard error is:

$$E \cong \frac{1}{\sqrt{2BT}} \left[1+\left[\frac{R_x^2(0)}{R_x^2(\tau)}\right]^{1/2}\right]$$

From this equation, an error, E, of two percent is predicted at =0 with a bandwidth, B = 100 hertz, and record length time, T = 20.48 seconds, typical values imployed in processing the data.

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[1] Langenthal, I. M., "Correlation and Probability Analysis," SAICOR Signals, TB 14, p.11.

V. RESULTS

A. ANALYSIS OF NORMAL PATIENTS

The relationship between the ECG signal and its correlation waveform depends upon spectral content. P, T, and QRS waves in the ECG signal will generate peaks in the correlation waveform. When the rhythm is constant the correlated peaks will have high definition - tall and narrow, whereas an ECG signal with a random beat or inconsistent frequency of repetition would display correlation peaks with low definition (deterioration of peaks to wide flat profiles). Recall the second mathematical property of the autocorrelation function is that $R_x(0)$ is the maximum value of the function. Disregarding $R_x(0)$, the peak that has the highest definition will normally correspond in frequency to the heart rate.

Crosscorrelation is used to determine the similarity between two signals. A common spectral content would be indicated by a particular peak in the crosscorrelation function. A complete lack of similarity between two signals would result in a crosscorrelation function devoid of any peaks.

Figure 7 contains the autocorrelation and crosscorrelation of a typical normal ECG. The characteristics of the ECG signal for Lead I which resulted in the associated correlation waveform are shown in Figures 7 (a) and (b). In this example the crosscorrelation of Lead I and II, Figure 7 (e), indicates a definite similarity between the two signals. Autocorrelation waveforms of ECG's taken from patients diagnosed normal by a cardiologist are shown in Figures 8 through 17.

After examination of the normal autocorrelation functions it is apparent that substantial variation in the correlations is to be expected and the success of the present technique depends on marked differences arising in the correlations of the ECG's of patients with known cardiac abnormalities.

B. ANALYSIS OF PATIENTS WITH ABNORMAL ELECTROCARDIOGRAPHS

The correlation of any patient who had an abnormal ECG (Figures 18 through 19) was analysed to determine the differences of that correlation from normal correlations. Abnormal rhythms, abnormal conduction, electrocardiographic position of the heart, and changes in the myocardium are discussed in this section.

1. Atrial Fibrillation

Atrial fibrillation is characterized by an irregular ventricular rhythm due to the A-V node transmitting impulses in a random fashion. Because the excitation waves move over the atrium at random rates, coordinated atrial contraction can not occur and individual P waves are hard to identify. Only a protion of these random excitation waves are transmitted to the ventricles. This randomness results in a complete absence of ventricular rhythm. As a result of the inconsistency of the heart beat, a low definition (lack of predominant peaks) in the correlation wave is to be expected.

Figure 20 shows the ECG and corresponding autocorrelation of a patient with atrial fibrillation. Note the irregular rhythm in each of the four ECG patterns and the low definition of the correlation waveform peaks. The more predominant peaks are generated by heart beats from 81 to 102 beats per minute. The less defined peak in Lead

I at 0.46 seconds corresponds to a heart beat of 125 beats per minute.

2. Mycardial Infarction

A myocardial infarction is a localized area of the mycordium which has become dead as a result of the tissue having been deprived of its blood supply because of a coronary obstruction. During the onset of a myocardial infarction the sequence of the ECG patterns change greatly from the initial occlusion of a coronary artery to the fully developed infarction. As the infarct heals, collateral blood vessels supply blood to the area surrounding the infarct and the ECG returns to a near normal pattern. Only during the coronary attack and phase of development of the myocardial infarction would there be any changes in the ECG pattern, therefore the correlated waveform should appear normal after recovery, as is typified by the record shown in Figure 21.

3. Mean Electrical Axis Deviation

The mean electrical axis is calculated from measurements of the QRS complex on Leads I and III. Since the mean electrical axis spatial vector is directed toward the posterior, rotation of the physical heart around its longitudinal axis produces large variations in the shape of each ECG lead. Right-axis deviation is present when the electrical axis is to the right of the range of the normal axis deviation and left-axis deviation is present when the electrical axis is to the left. This abnormality does not affect the rhythm of the heart, as can be observed in Figure 19 (a). This patient has a left axis deviation.

4. Sinus Tachycardia and Sinus Bradycardia

When the sinus node creates a rate greater than 100 beats per minute the rhythm is called sinus tachycardia. When the rate is less than 60 beats per minute the rhythm is called sinus bradycardia. The rhythm is constant for both of these abnormalities, therefore the correlation should be normal. Figure 18 (a) shows the correltation of a patient with sinus tachycardia. The correlation is normal computed to be 123 beats per minute - sinus tachycardia.

Patient 33, Figures 18 (e) and (f), has a cardiac rate of less than 60 beats per minute, however, the additional abnormality of ventricular contractions, discussed below, dominates the correlation. No other patients were diagnosed to have sinus bradycardia.

5. Premature Ventricular Contractions

When the S-A node rate is low or weak some other location in the heart (ectopic focus) may begin to discharge conduction impulses. When the ectopic foucs is in the ventricular myocardium, distorted QRS complexes of widely varying form and lack of P waves are the observable effects of this ectopic arrhythmia. When there are multiple ectopic foci or a random pulsing single ectopic focus, the ECG waveforms do not have a fixed relationship to the preceding waveform. Thus, the correlation waveform should have low definition. The ECG waveform of patient 33, Figure 18 (f) is not consistent and the associated correlation function shows the expected low-definition waveform.

6. Block of the Purkinje Fibers (Bundle Branch Block)

Blockage of the Purkinje fibers requires that the cardiac impulse be conducted by the ventricular muscle instead, thereby decreasing the rate of impulse conduction to approximately one-third

normal. This delay increases the duration of the QRS complex to approximately 0.16 seconds (duration greater than 0.08 seconds is considered abnormal). When the block is on only one side of the ventricle the cardiac impulse is delayed upon reaching the block causing the electrical axis of the heart to shift. Thus a left axis deviation is normally associated with a left bundle branch block. Although the QRS complex is distorted it is still consistent in rhythm, therefore the correlation should be normal and show a high degree of definition.

Figure A-12 (a) and (b) show the autocorrelation and ECG of a patient with left bundle branch block. The ECG waveform is definitely distorted and the associated correlation shows the high definition of a normal patient.

7. Patients with Multiple Abnormalities

Patient 33, Figure 22, has a history of sinus bradycardia and associated premature ventricular contractions. A pacemaker was emplaced one month before the recorded ECG. The pacemaker is partially inhibited and does not maintain a constant rate, therefore its effect on the correlation should be minimal. The effect of sinus bradycardia on the correlation, that of a heart beat less than 60 beats per minute, would be overridden by the random effect of the premature ventricular contractions. Thus the correlation should show low definition peaks with the predominant peak (if there is one) corresponding to a heart beat of less than 60 beats per minute, as is illustrated in Figure 22.

Patient 31, Figure 23, has an abnormal ECG due to left bundle branch block, left axis deviation, and sinus tachycardia. The left axis deviation and the left bundle branch block have no effect on the regularity of the rhythm. Sinus tachycardia denotes a faster than

normal rhythm, therefore the correlation of the patient's ECG should have high definition representative of a normal patient, which is the case shown in Figure 23.

C. EXPERIMENTAL UNCERTAINTY

The expected experimental uncertainty was estimated to be 2% as indicated in Section IV. To determine the validity of this theoretical uncertainty different record lengths (length of recorded ECG signal) were autocorrelated. The results shown in Figure 24 indicate the actual experimental error to be less than one percent for the selected experimental record length of twenty seconds. The results for each record length are:

Record length (sec)	Sumations	Percent Error
5.12	512	1.8
10.24	1024	1.2
20.48	2048	0.9

It was assumed the correlated ECG signals of relatively weak magnitude would have characteristics similar to white noise (steadily decaying correlation function). Figure 19 (e) shows such an expected example. Therefore a test was devised to determine the poorest usable signal to noise ratio (s/n) where the correlated waveform indicated noticeable deterioration. Figure 25 depicts the autocorrelation of an ECG signal as it is progressively attenuated both with a 10mV varying frequency sinusoidal signal imposed as the noise signal and without the noise signal imposed. With no imposed noise signal, there was no detectable deterioration of the correlated waveform even when the ECG signal peak to peak voltage was attenuated to less than 10 mV. With the imposed noise signal, deterioration was detected at a s/n of 3/1 and complete deterioration was detected at 2/1. Therefore a signal of

peak to peak voltage greater than 10 mV with a s/n greater than 3/1 could be reliably correlated with the present system.

Some of the correlated waveform peaks could not be related to the characteristics of the associated ECG signal on several of the patients analyzed. The correlation of Lead I of patient 6 is an example (Fig. 26 (a)). The expected correlation was a peak at 67.5 beats/min. with low definition secondary peaks due to the low amplitude of the P and T waves. The autocorrelation, however, had high definition peaks at 270, 135, 90, and 67.5 cycles/minute as can be seen in Figure 26 (a). Investigation as to the quality of the tape recorded signal revealed a spurious signal which can be seen when Figure 26 (d) is compared with Figure 26 (e). This spurious signal was traced to a 27 kHz voltage controlled bias oscillator in the Telex instrumentation tape recorder. No convenient means was discovered for the suppression of the unwanted signal.

VI. SUMMARY AND CONCLUSIONS

Because of its usefulness in enhancing the information contained in periodic signals, correlation analysis appeared promising as a means of simplifying the interpretation of electrocardiographic records. An investigation employing such techniques has been made into the ECG's of thirty-six patients, both normal and with specific diagnosed cardiac disorders.

Thirty of the thirty-six patients analyzed were diagnosed as normal by the cardiologist. By being selective in eliminating the records that did not meet the s/n criteria, every one of the normal patients had a predictable correlation. However, no unique characteristic of normal correlation waveforms could be isolated other than the high definition of the correlation waveform.

Arrhythmias, where there is a random rhythm or change in the ECG waveform from one beat to another, can be detected by correlation. The resultant correlation will be of low definition as shown in Figures 18 (e) and 19 (e). It does not matter greatly which electrocardiographic lead is recorded when correlation is performed to determine arrhythmias, since arrhythmias detectable by correlation depend on the time relationships between the different waves of the cardiac cycle. Identification of the specific abnormality can not be accomplished by either the autocorrelation or the crosscorrelation function, therefore the correlation function does not appear to be useful in diagnosis. In general abnormalities such as electrical axis deviation, sinus tachycardia, and

sinus bradycardia do not involve arrhythmic behavior and the correlation technique is not useful in detecting such abnormalities.

Although correlation analysis can not determine the specific arrhythmia, the correlation function can detect changing cardiac waveform whether that change is due to cardiac myopathies or arrhythmia. Thus the proposed method might be used in an intensive care unit of a hospital where the primary requirement is detection of deviant cardiac behavior as rapidly as possible.

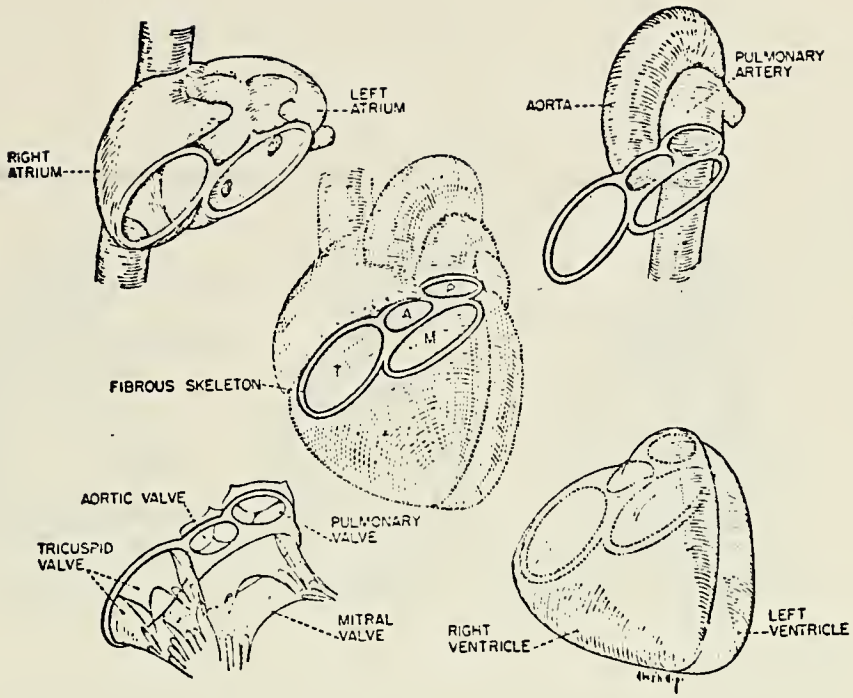


Figure 1. Anatomic Components of the Heart.

The fibrous skeleton of the heart consists of four valve rings - aortic, mitral, tricuspid, and pulmonary. To these rings are fastened the two major arterial trunks (aorta and the pulmonary artery) and all four cardiac chambers (left and right ventricle, and left and right atrium).

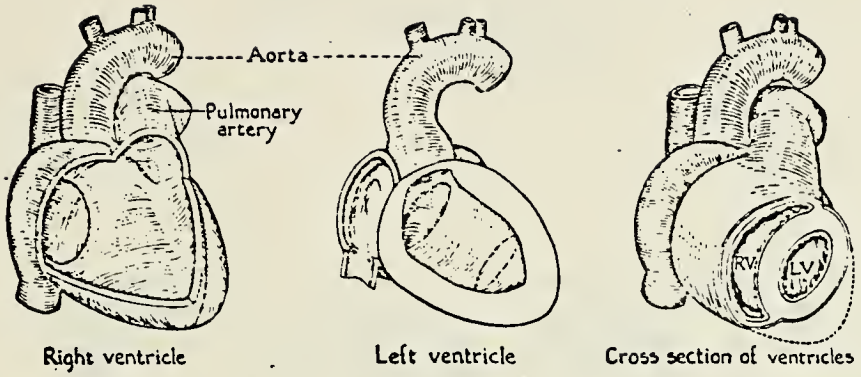
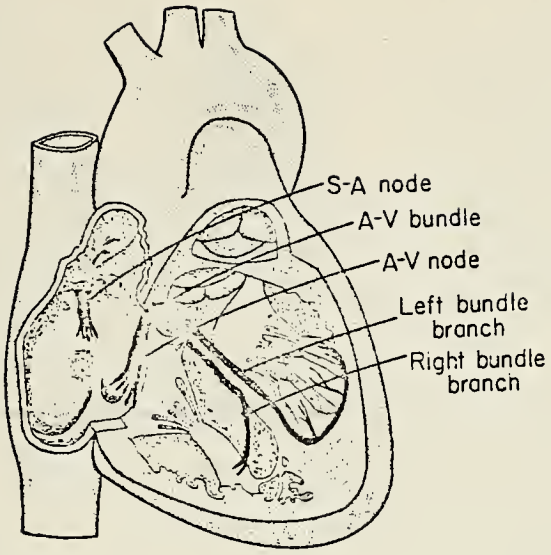
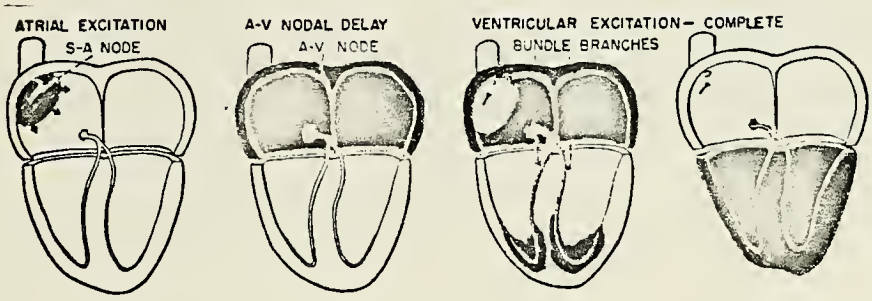


Figure 2. Configuration of the Ventricular Chambers.

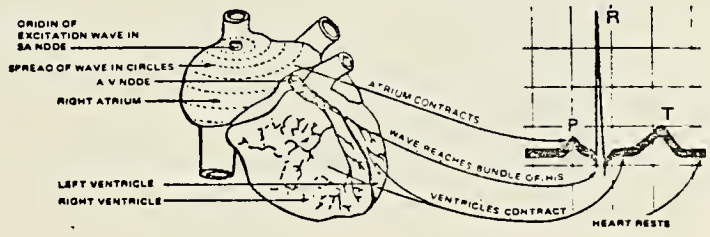
The right ventricular cavity is enclosed by the convex interventricular septum and the concave free wall, which may be considered a segment of a very large sphere. The myocardial bundles in the left ventricle are thicker than those in the right ventricular cavity.



(a) Conduction System.

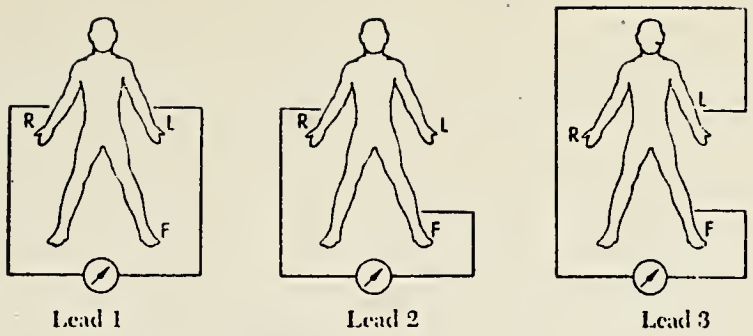


(b) Sequence of Cardiac Excitation

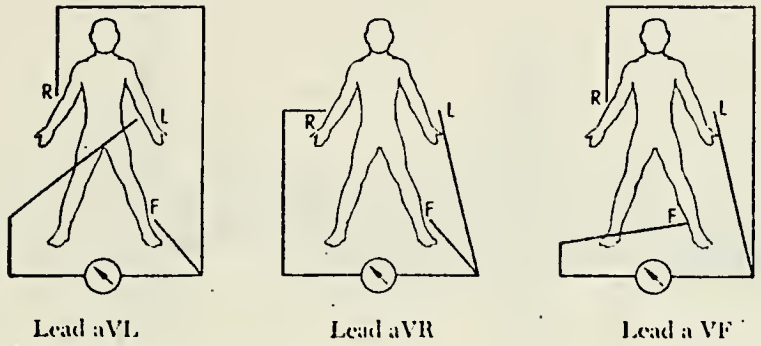


(c) Spread of Excitation Wave.

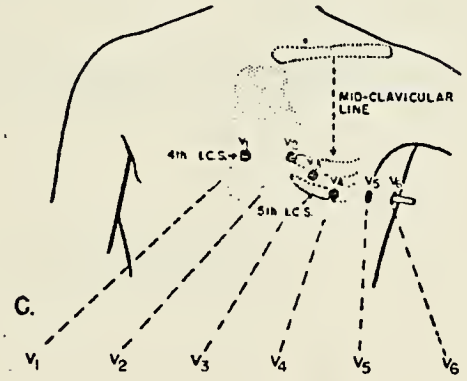
Figure 3. Conduction System of the Heart.



(a) Three Bipolar Standard Leads.



(b) Three Augmented Unipolar Leads.



(c) Six Unipolar Precordial Leads.

Figure 4. Normal Twelve Electrode Positions.

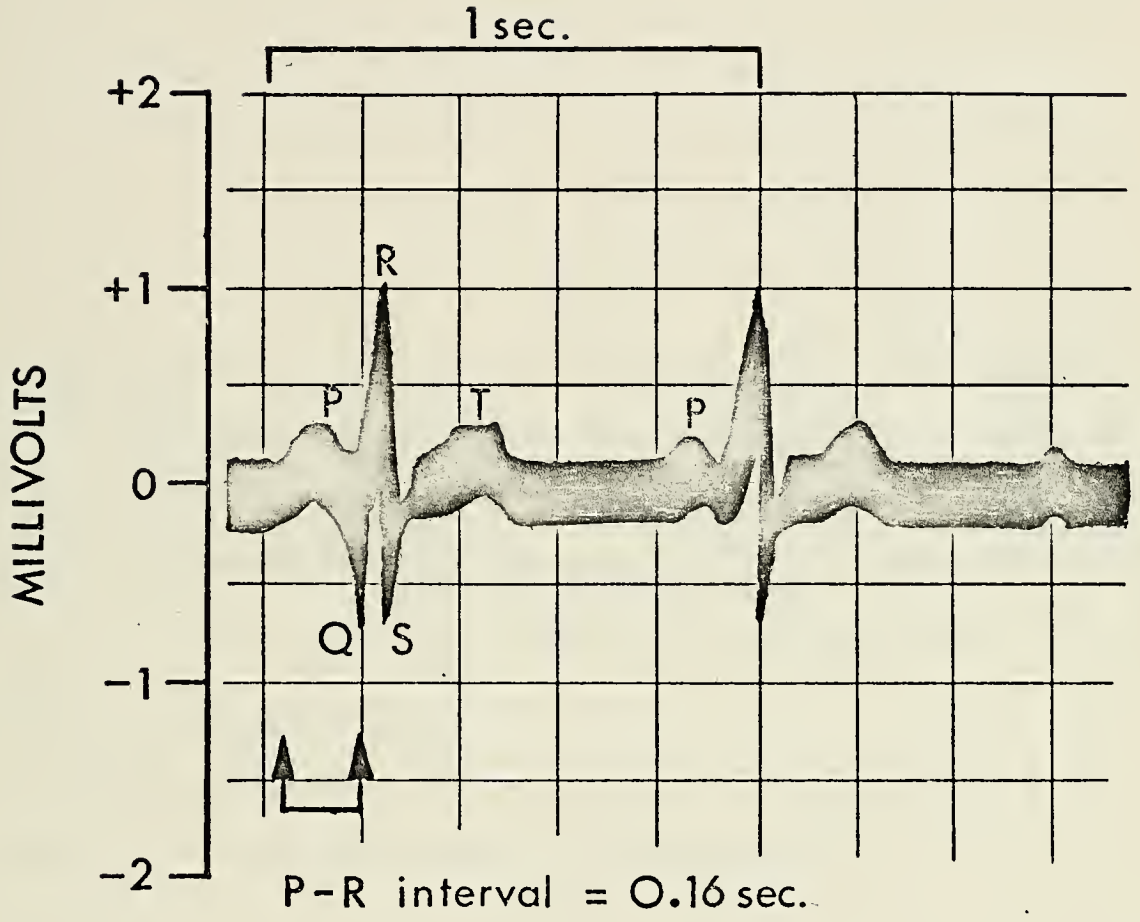


Figure 5. The Normal Electrocardiogram.

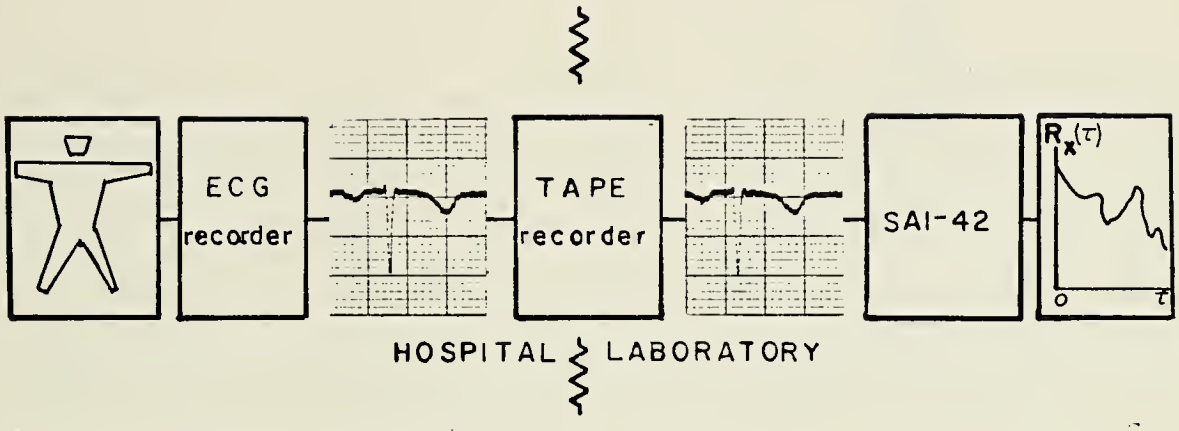
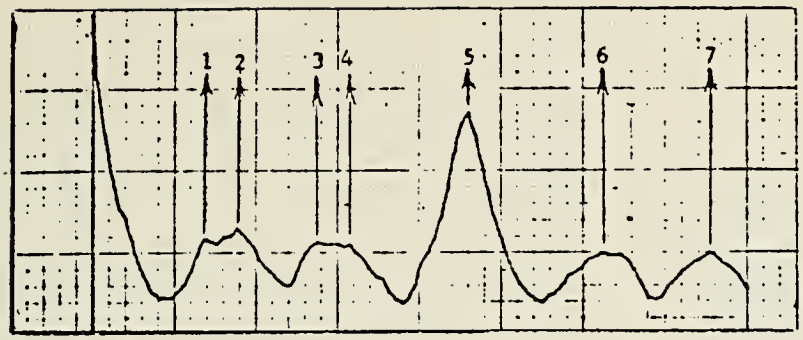
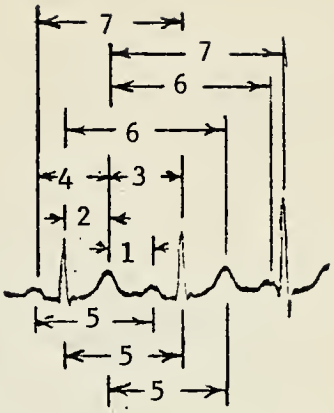


Figure 6. Experimental Setup.

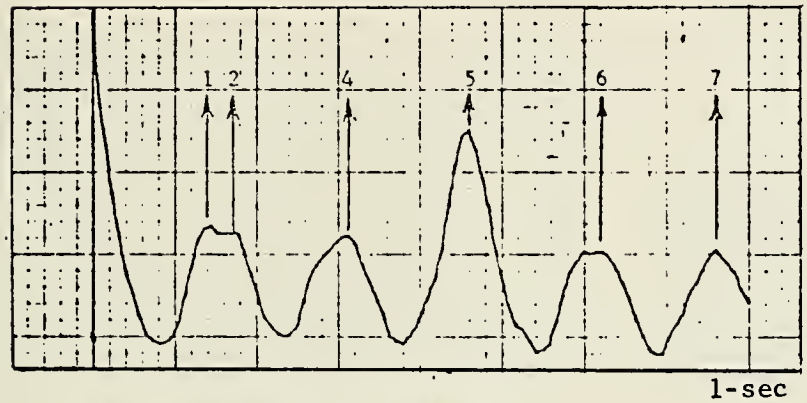
Electrocardiographic signals were recorded in the hospital and analysed in the NPS Aeronautical Engineering laboratory.



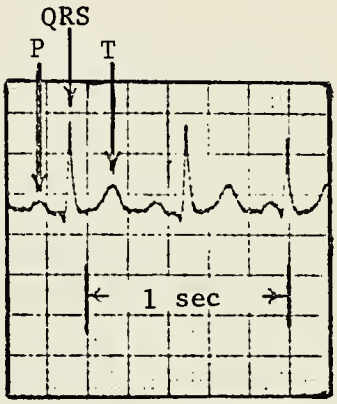
(a) Autocorrelation of Lead I.



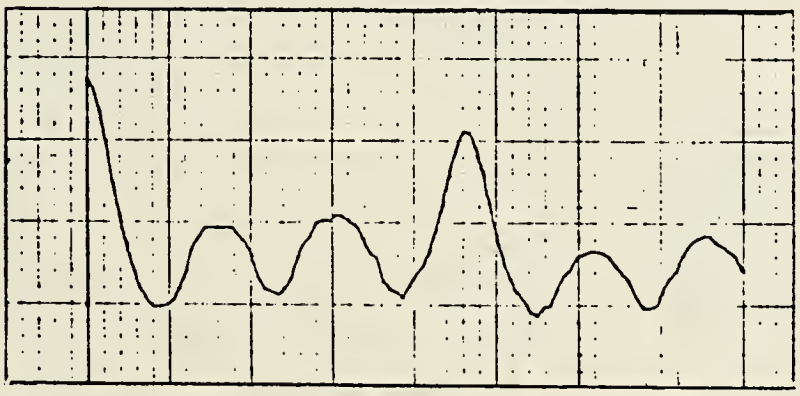
(b) ECG Lead I.



(c) Autocorrelation of Lead II.



(d) ECG Lead II.

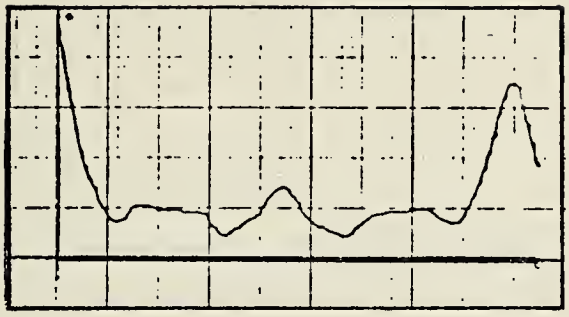


(e) Crosscorrelation of Lead I and Lead II.

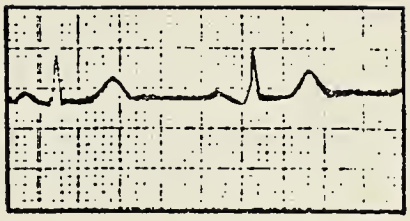
LEGEND

1. T P wave
2. QRS T wave
3. T QRS complex
4. P T wave
5. P P; T T; QRS QRS
6. QRS 2nd T wave; T 2nd P wave
7. P 2nd T wave; T 2nd QRS

Figure 7. Autocorrelation and Crosscorrelation of Normal Patient.

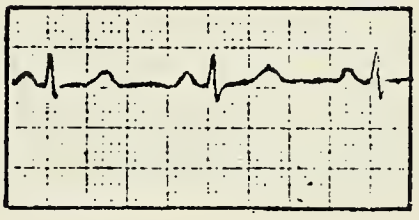


(a) Patient 1: Autocorrelation: AVF



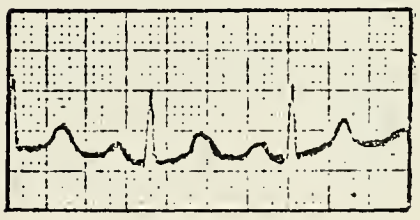
(b) Patient 1: ECG - AVF

(c) Patient 2: Autocorrelation - Lead II



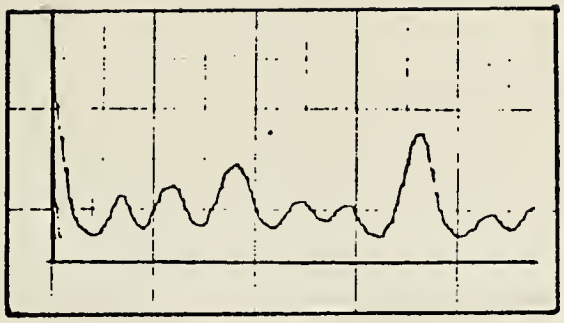
(d) Patient 2: ECG - Lead II

(e) Patient 3: Autocorrelation - Lead II

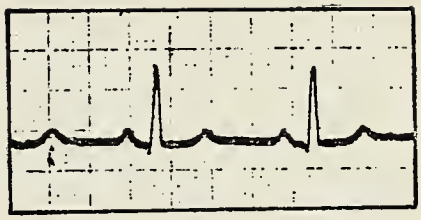


(f) Patient 3: ECG - Lead II

Figure 8. Autocorrelation and ECG Graphs of Patients 1-3.

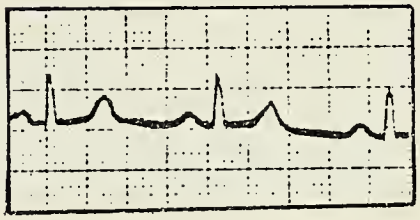


(a) Patient 4: Autocorrelation - Lead II



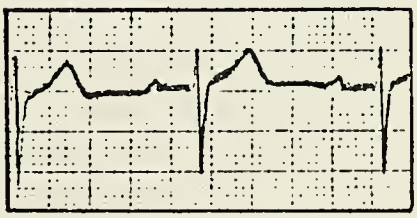
(b) Patient 4: ECG - Lead II

(c) Patient 5: Autocorrelation - Lead II



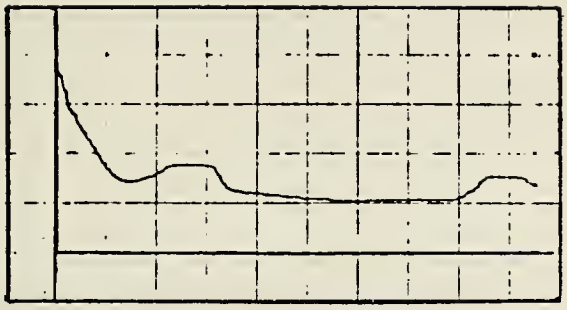
(d) Patient 5: ECG - Lead II

(e) Patient 6: Autocorrelation - Lead V3



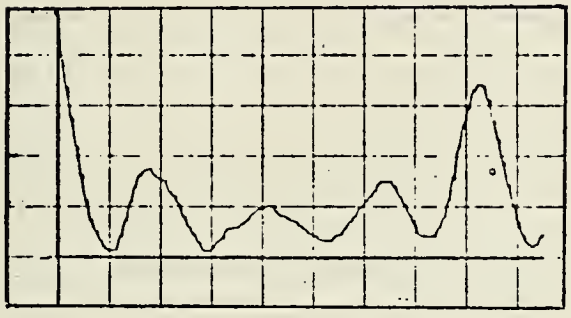
(f) Patient 6: ECG - Lead V3

Figure 9. Autocorrelation and ECG Graphs of Patients 4-6.

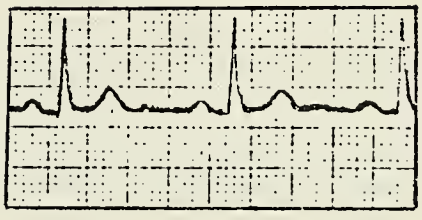


(a) Patient 7: Autocorrelation - Lead V4

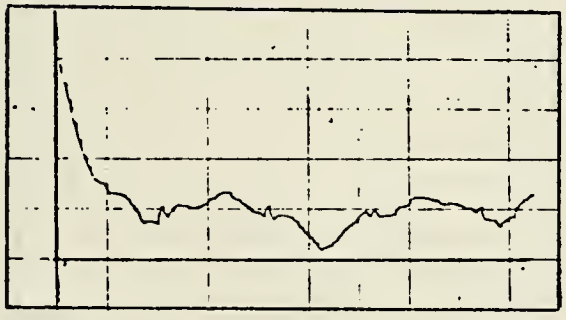
(b) Patient 7: ECG - Lead V4



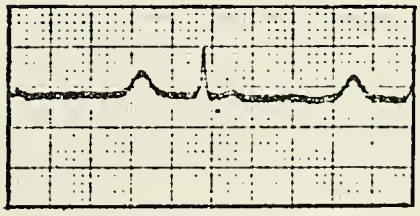
(c) Patient 8: Autocorrelation - Lead II



(d) Patient 8: ECG - Lead II

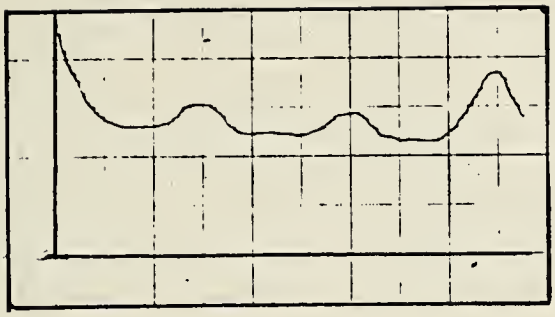


(e) Patient 9: Autocorrelation - Lead AVR

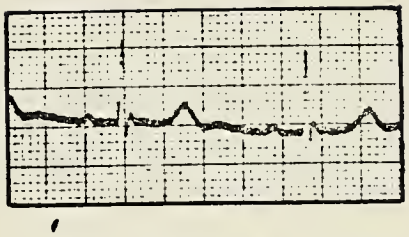


(f) Patient 9: ECG - Lead AVR

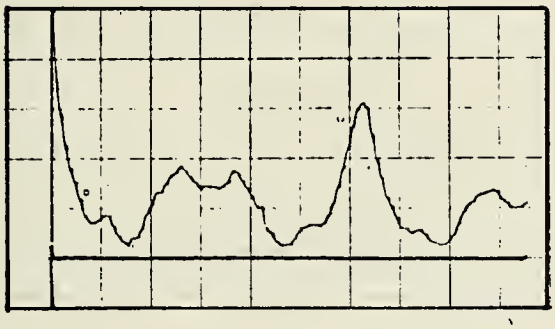
Figure 10. Autocorrelation and ECG Graphs of Patients 7-9.



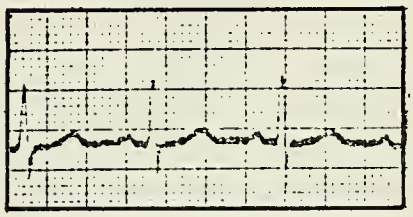
(a) Patient 10: Autocorrelation - Lead V3



(b) Patient 10: ECG - Lead V3

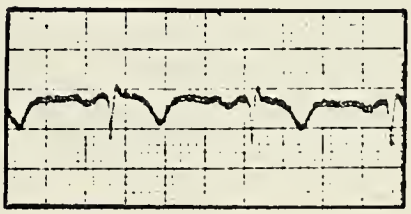


(c) Patient 11: Autocorrelation - Lead I



(d) Patient 11: ECG - Lead I

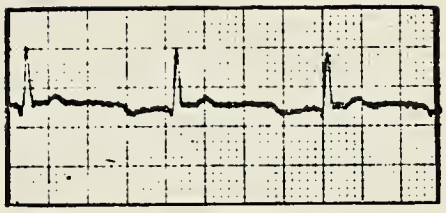
(e) Patient 12: Autocorrelation - Lead AVR



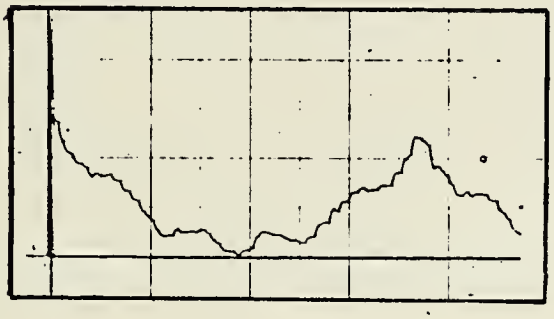
(f) Patient 12: ECG - Lead AVR

Figure 11. Autocorrelation and ECG Graphs of Patients 10-12.

(a) Patient 13: Autocorrelation - Lead AVL



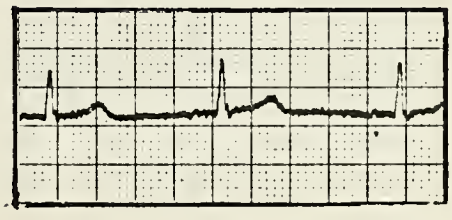
(b) Patient 13: ECG - Lead AVL



(c) Patient 14: Autocorrelation - Lead AVR

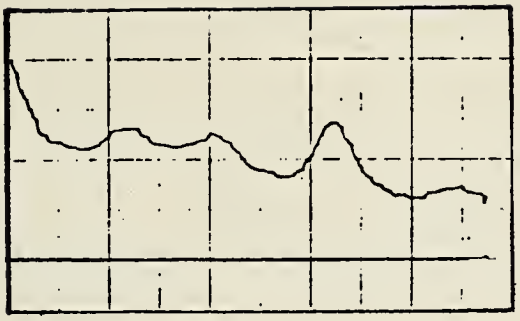
(d) Patient 14: ECG - Lead AVR

(e) Patient 15: Autocorrelation - Lead AVL

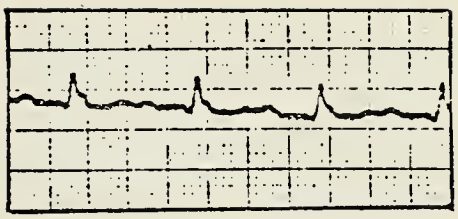


(f) Patient 15: ECG - Lead AVL

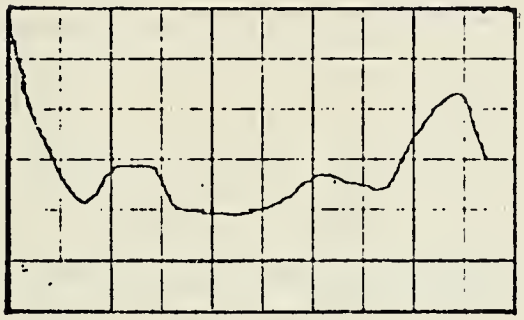
Figure 12. Autocorrelation and ECG Graphs of Patients 13-15.



(a) Patient 16: Autocorrelation - Lead AVF



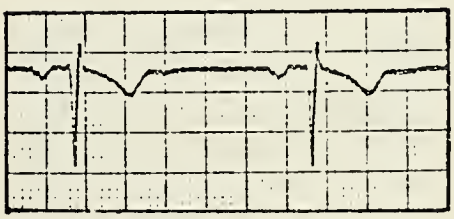
(b) Patient 16: ECG - Lead AVF



(c) Patient 17: Autocorrelation - Lead V5

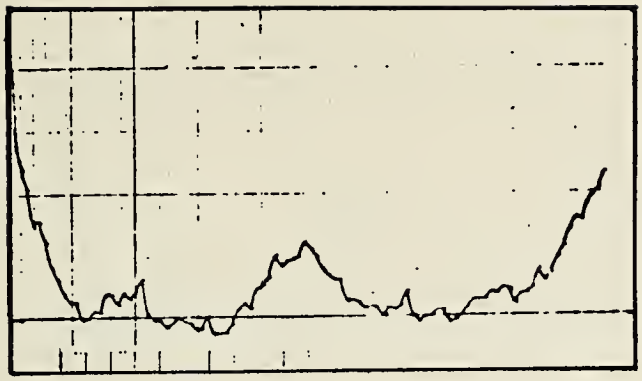
(d) Patient 17: ECG - Lead V5

(e) Patient 18: Autocorrelation - Lead AVR



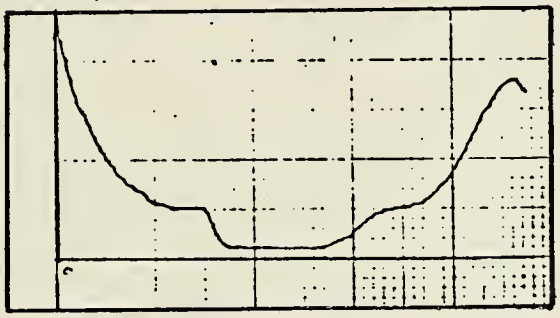
(f) Patient 18: ECG - Lead AVR

Figure 13. Autocorrelation and ECG Graphs of Patients 16-18.



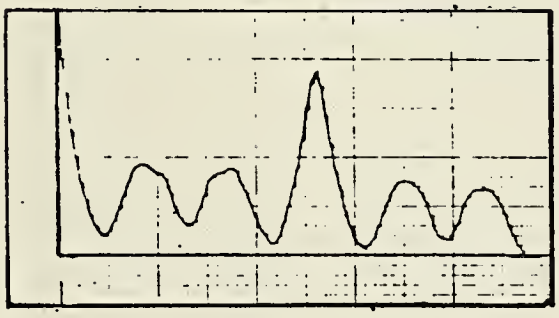
(a) Patient 19: Autocorrelation - Lead AVR

(b) Patient 19: ECG - Lead AVR

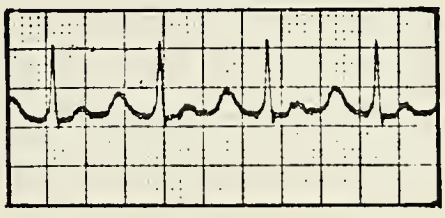


(c) Patient 20: Autocorrelation - Lead V4

(d) Patient 20: ECG - Lead V4

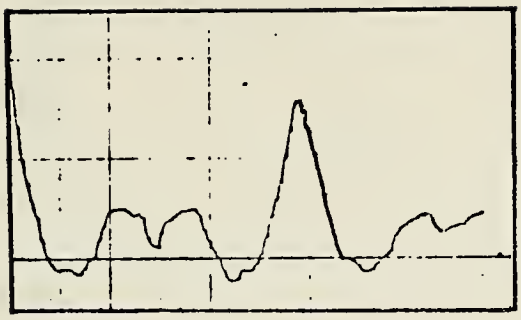


(e) Patient 21: Autocorrelation - Lead AVR

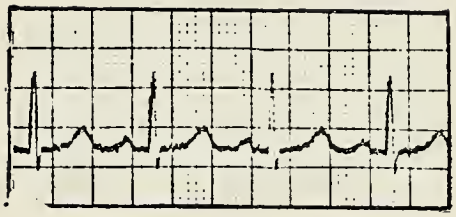


(f) Patient 21: ECG - Lead AVR

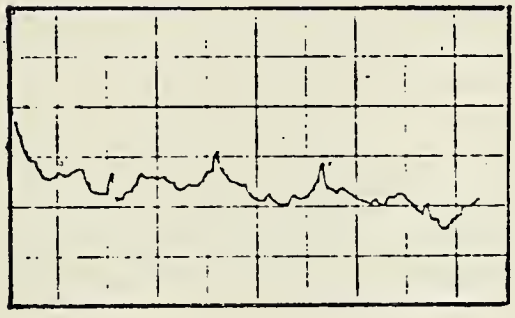
Figure 14. Autocorrelation and ECG Graphs of Patients 19-21.



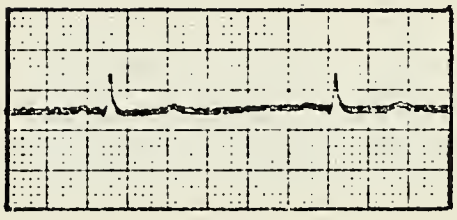
(a) Patient 22: Autocorrelation - Lead I



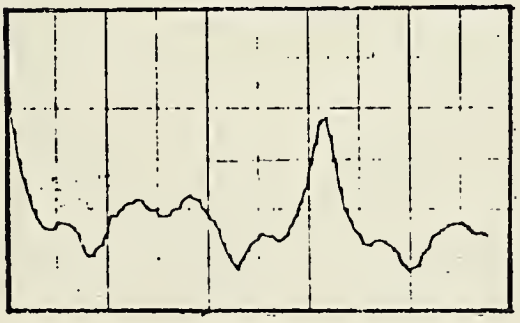
(b) Patient 22: ECG - Lead I



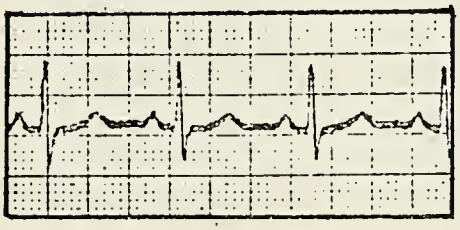
(c) Patient 23: Autocorrelation - Lead AVL



(d) Patient 23: ECG - Lead AVL

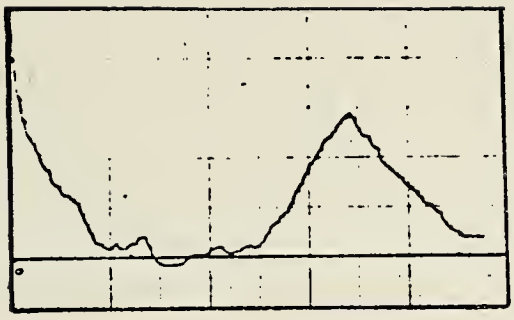


(e) Patient 24: Autocorrelation - Lead II

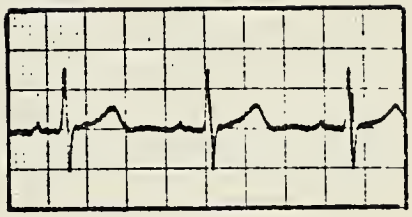


(f) Patient 24: ECG - Lead II

Figure 15. Autocorrelation and ECG Graphs of Patients 22-24.

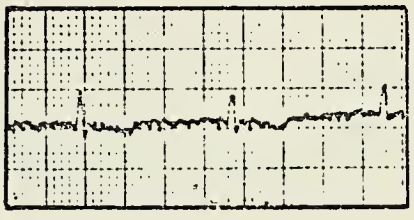


(a) Patient 25: Autocorrelation - Lead I



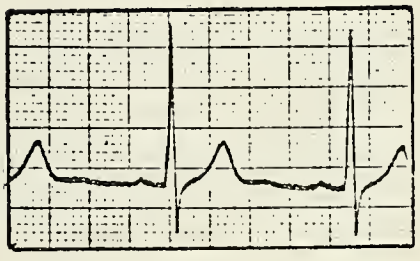
(b) Patient 25: ECG - Lead I

(c) Patient 26: Autocorrelation - Lead AVL



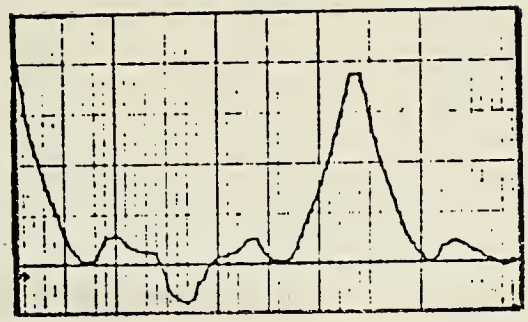
(d) Patient 26: ECG - Lead AVL

(e) Patient 27: Autocorrelation - Lead V4

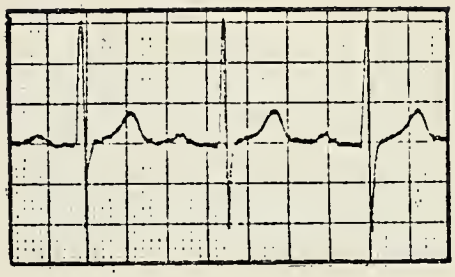


(f) Patient 27: ECG - Lead V4

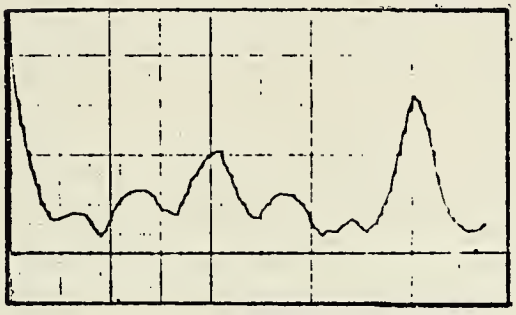
Figure 16. Autocorrelation and ECG Graphs of Patients 25-27.



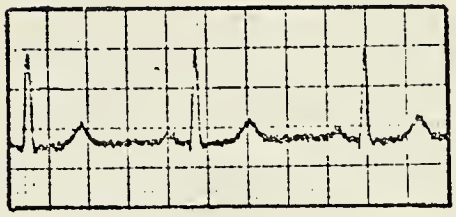
(a) Patient 28: Autocorrelation - Lead V4



(b) Patient 28: ECG - Lead V4

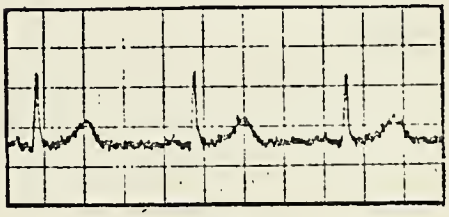


(c) Patient 29: Autocorrelation - Lead I



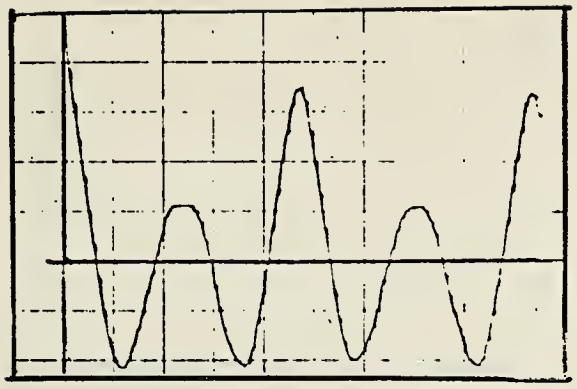
(d) Patient 29: ECG - Lead I

(e) Patient 30: Autocorrelation - Lead I

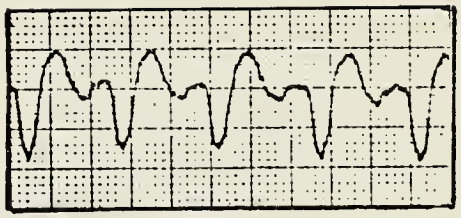


(f) Patient 30: ECG - Lead I

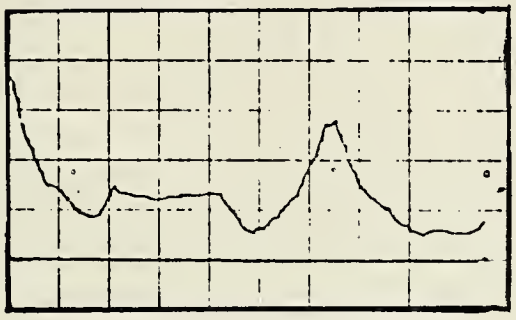
Figure 17. Autocorrelation and ECG Graphs of Patients 28-30.



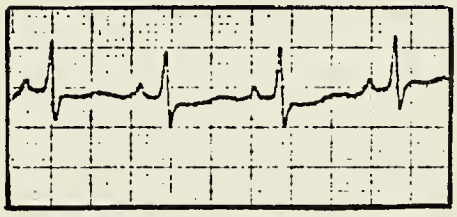
(a) Patient 31: Autocorrelation - Lead AVR



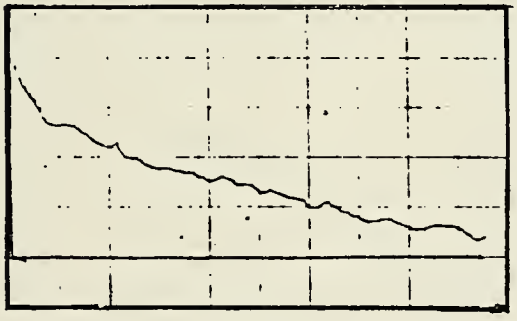
(b) Patient 31: ECG - Lead AVR



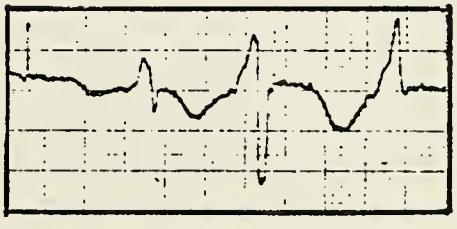
(c) Patient 32: Autocorrelation - Lead I



(d) Patient 32: ECG - Lead I



(e) Patient 33: Autocorrelation - Lead I



(f) Patient 33: ECG - Lead I

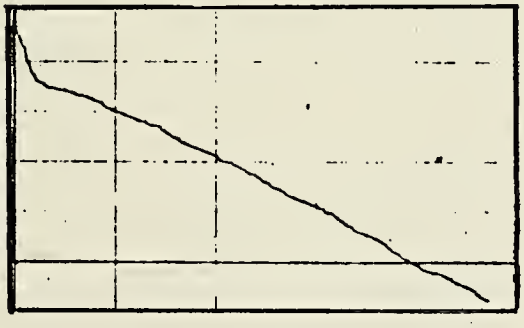
Figure 18. Autocorrelation and ECG Graphs of Patients 31-33.

(a) Patient 34: Autocorrelation - Lead AVL

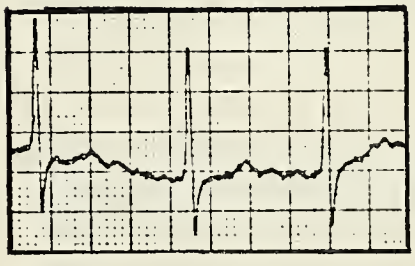
(b) Patient 34: ECG - Lead AVL

(c) Patient 35: Autocorrelation - Lead AVR

(d) Patient 35: ECG - Lead AVR



(e) Patient 36: Autocorrelation - Lead V5



(f) Patient 36: ECG - Lead V5

Figure 19. Autocorrelation and ECG Graphs of Patients 34-36.

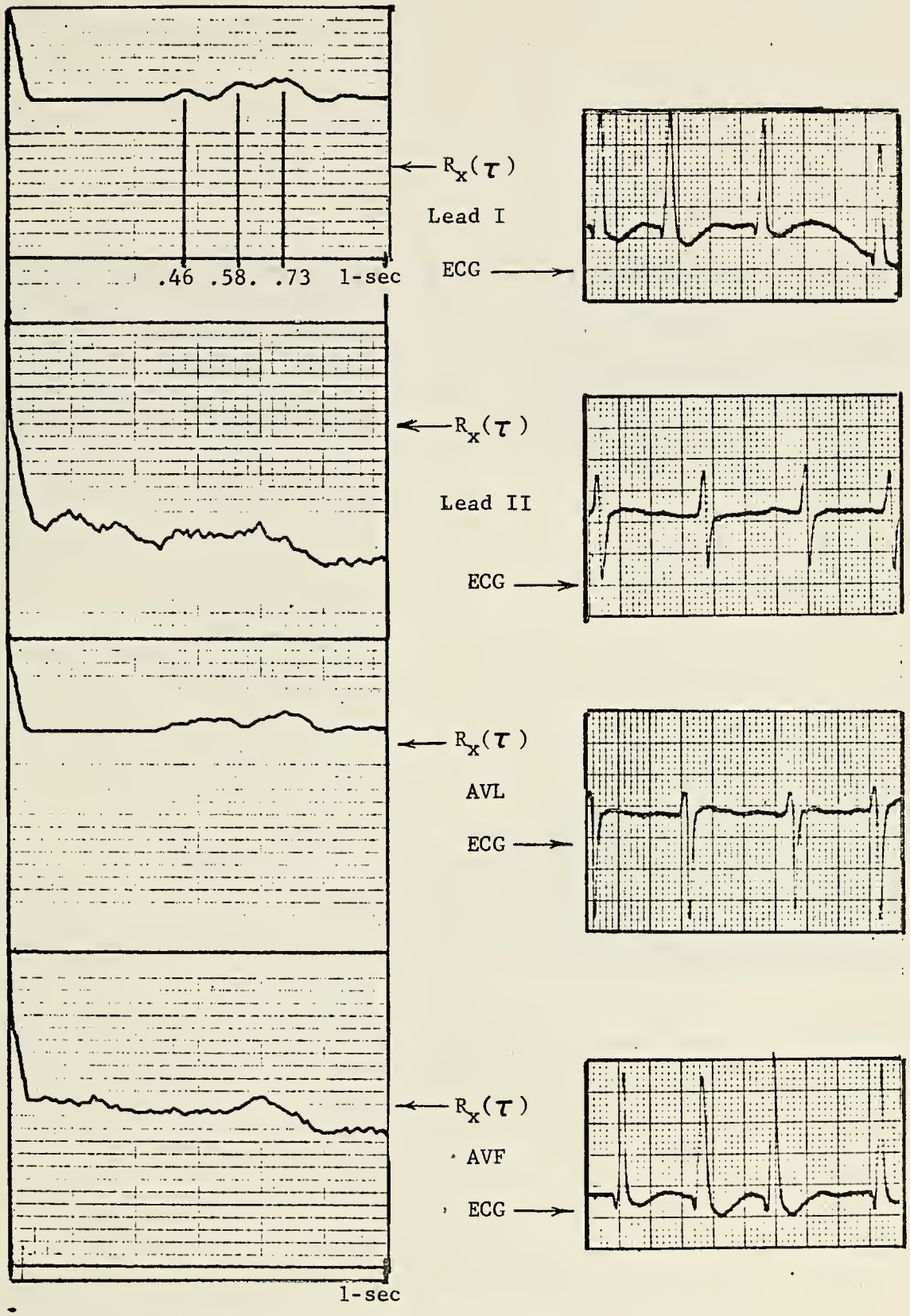


Figure 20. Patient 36: Atrial Fibrillation.

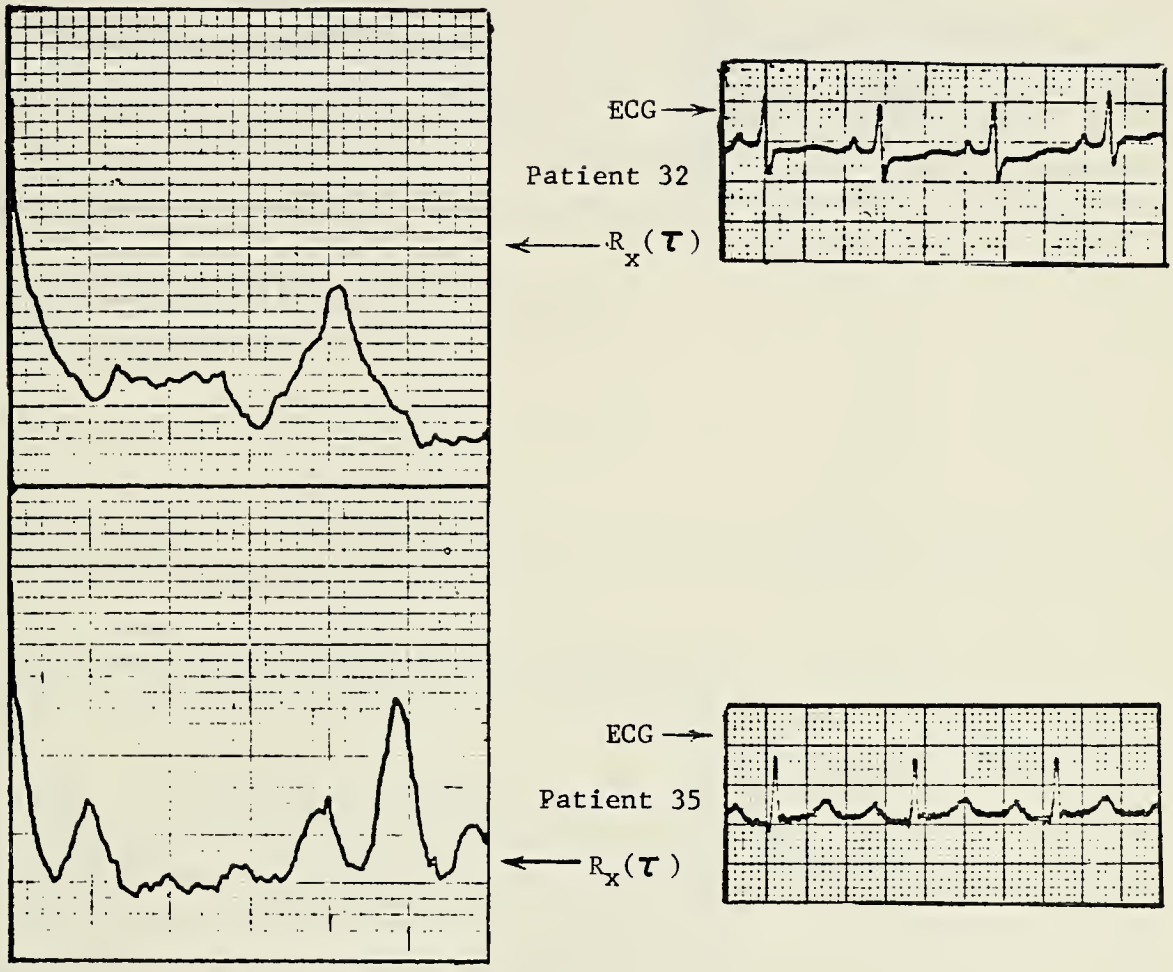
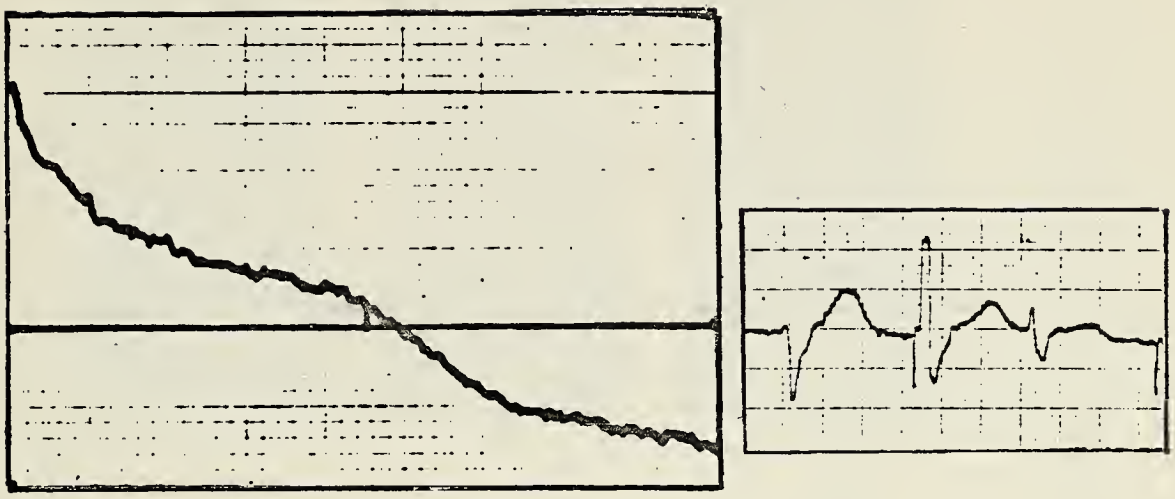
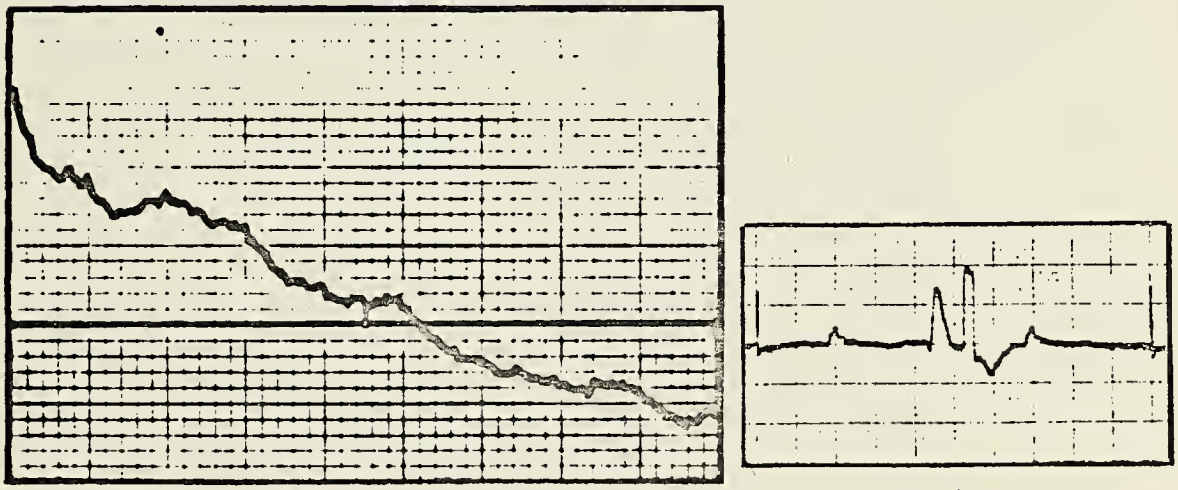


Figure 21. Patients with myocardial infarction.



(a) Autocorrelation and ECG of Lead I



(b) Autocorrelation and ECG of Lead III

Figure 22. Patient 33: Sinus Bradycardia and PVC.

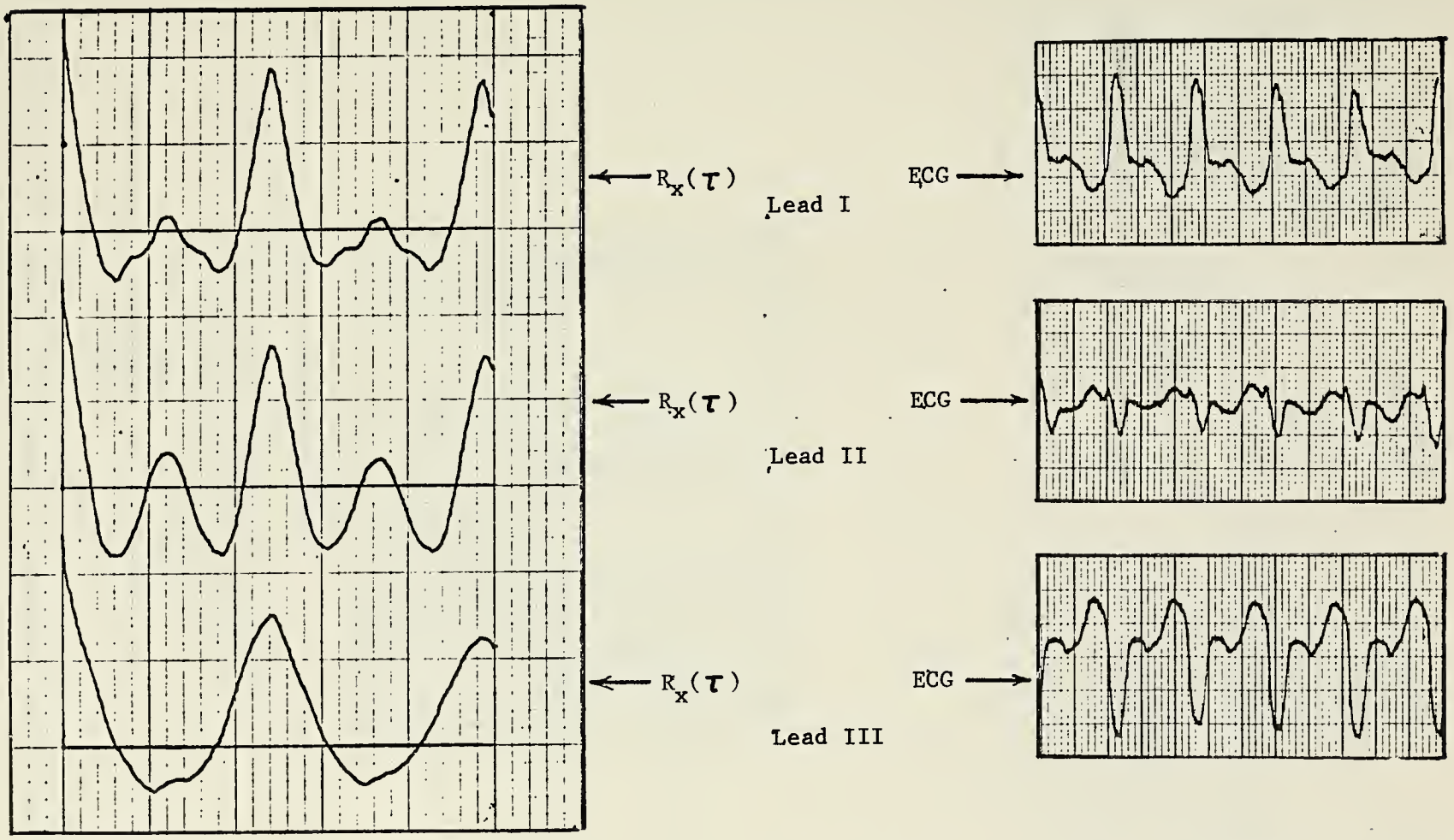
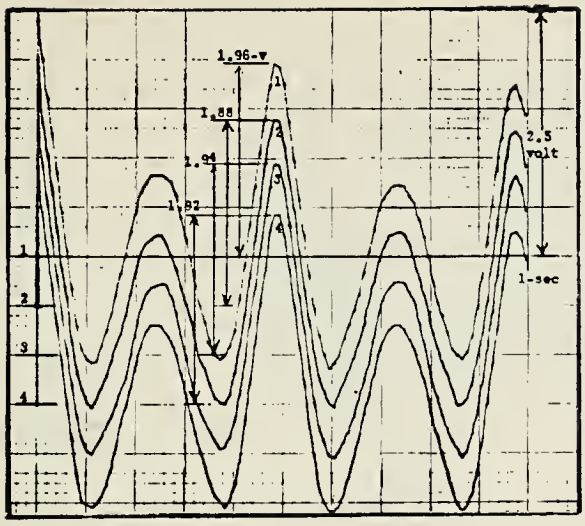
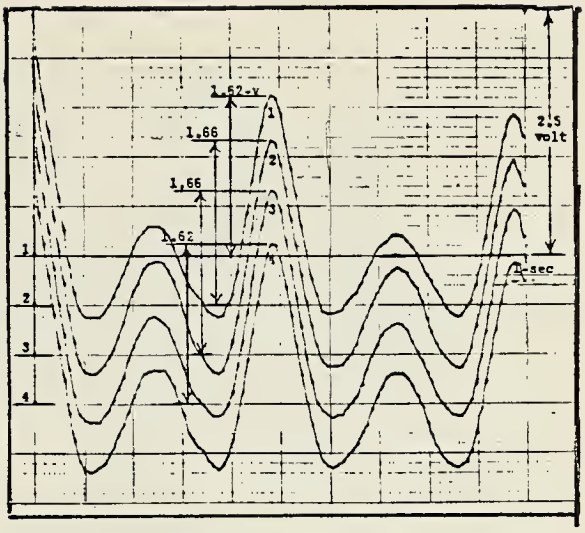


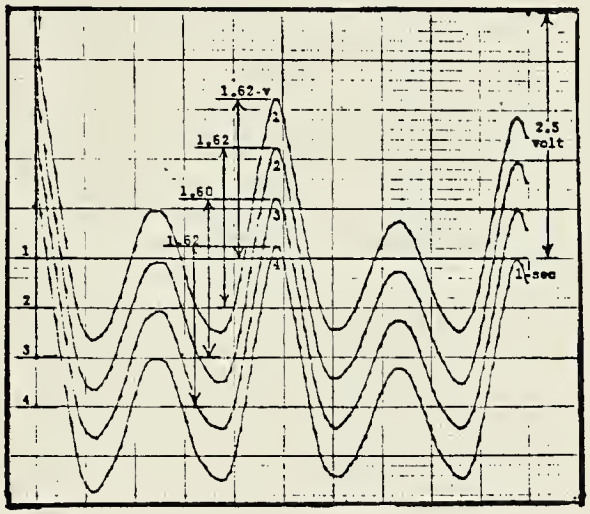
Figure 23. Patient 31: Left Bundle Branch Block, Left Axis Deviation, and Sinus Tachycardia.



(a) 512 Summations.



(b) 1024 Summations.

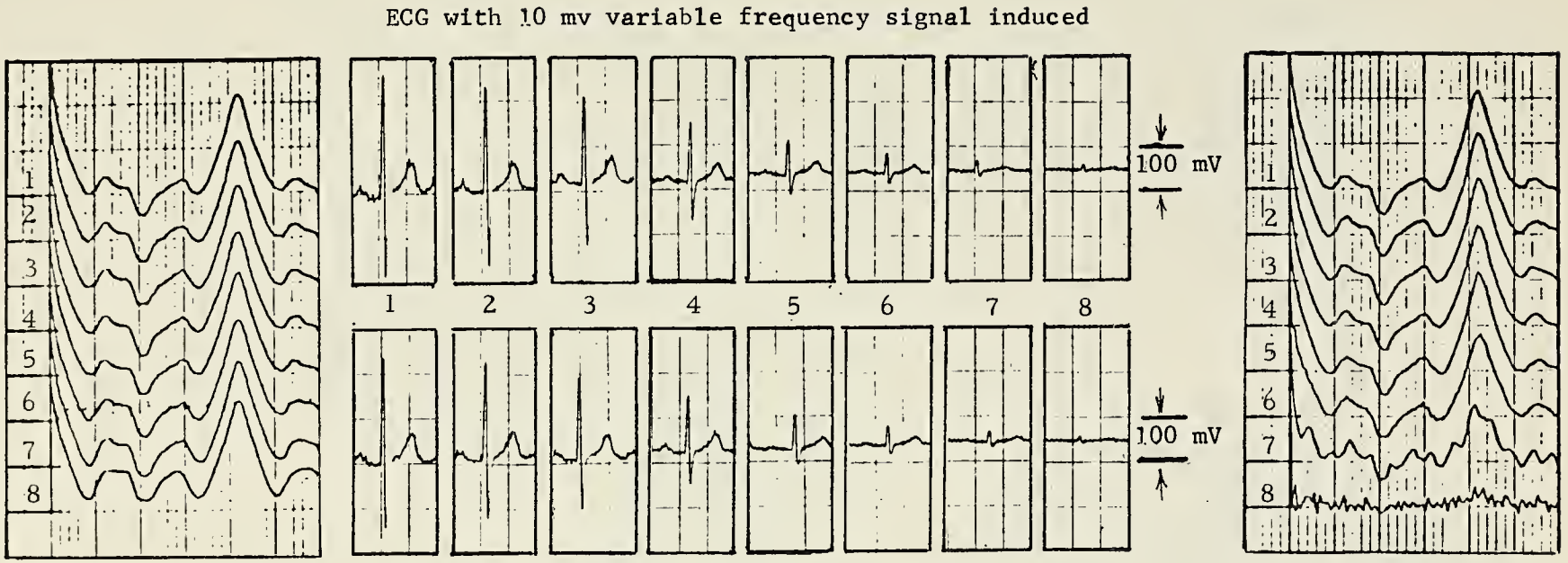


(c) 2048 Summations.

Experimental Error Determination

Summations	Percent Error
512	1.8
1024	1.2
2048	0.9

Figure 24. Sample Error with Different Summation Quantities.

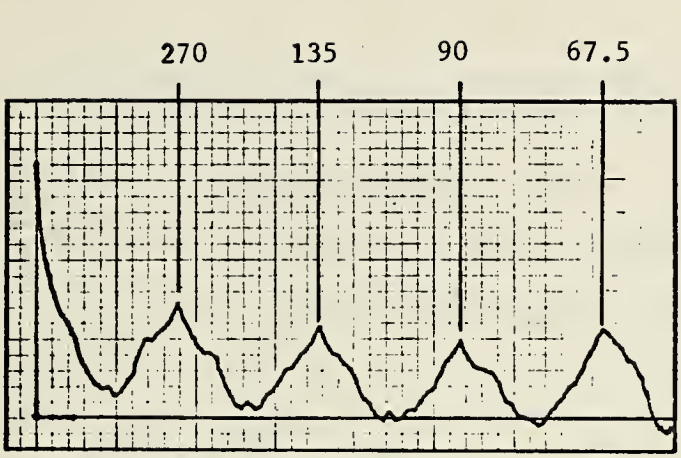


Correlated signal no induced noise.

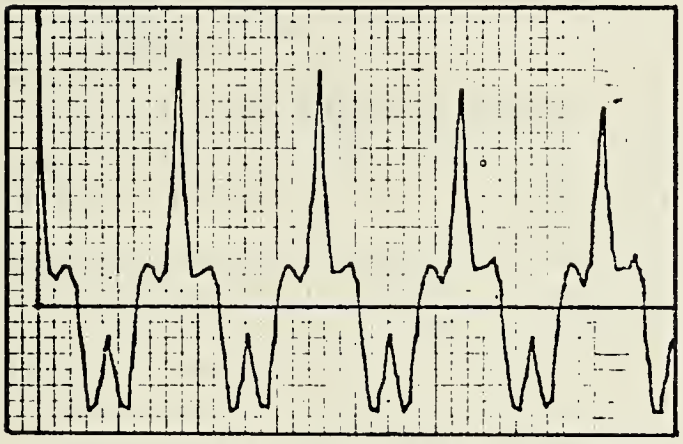
ECG with no variable sinusoidal signal induced

Correlated signal 10mV variable frequency sinusoidal signal induced.

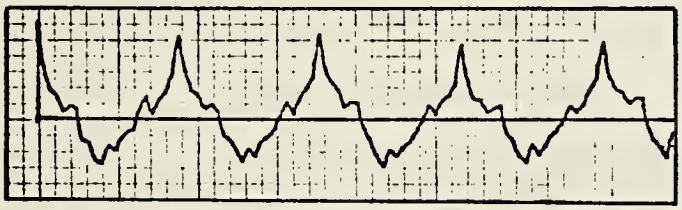
Figure 25. Determination of Threshold Signal to Noise Ratio.



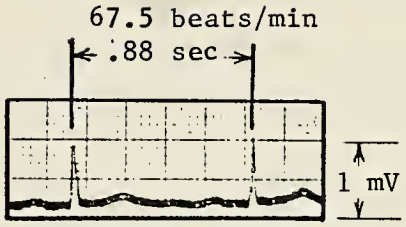
(a) Autocorrelation Lead I.



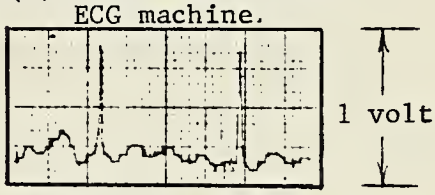
(b) Autocorrelation of blank channel.



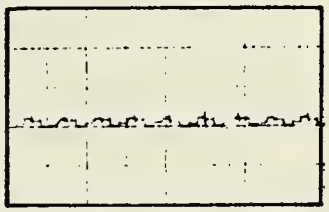
(c) Crosscorrelation of Lead I and blank channel.



(d) ECG- Lead I from ECG machine.



(e) ECG- Lead I from taperecorder.



(f) Noise from blank channel.

Figure 26. Patient 6: 27 kHz noise spikes.

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13. ABSTRACT

A method for the interpretation of electrocardiographic waveforms by correlation analysis has been investigated. Both the autocorrelation function and the crosscorrelation function are employed in the analysis. The electrocardiographic signals of thirty-six patients, including normal persons and those with specific diagnosed coronary pathology, have been analyzed. Electrocardiographic signs of abnormal heart rate, rhythm, and conduction patterns may be detected by correlation methods; however, the correlated wave form does not appear to be useful in determining the specific abnormality.

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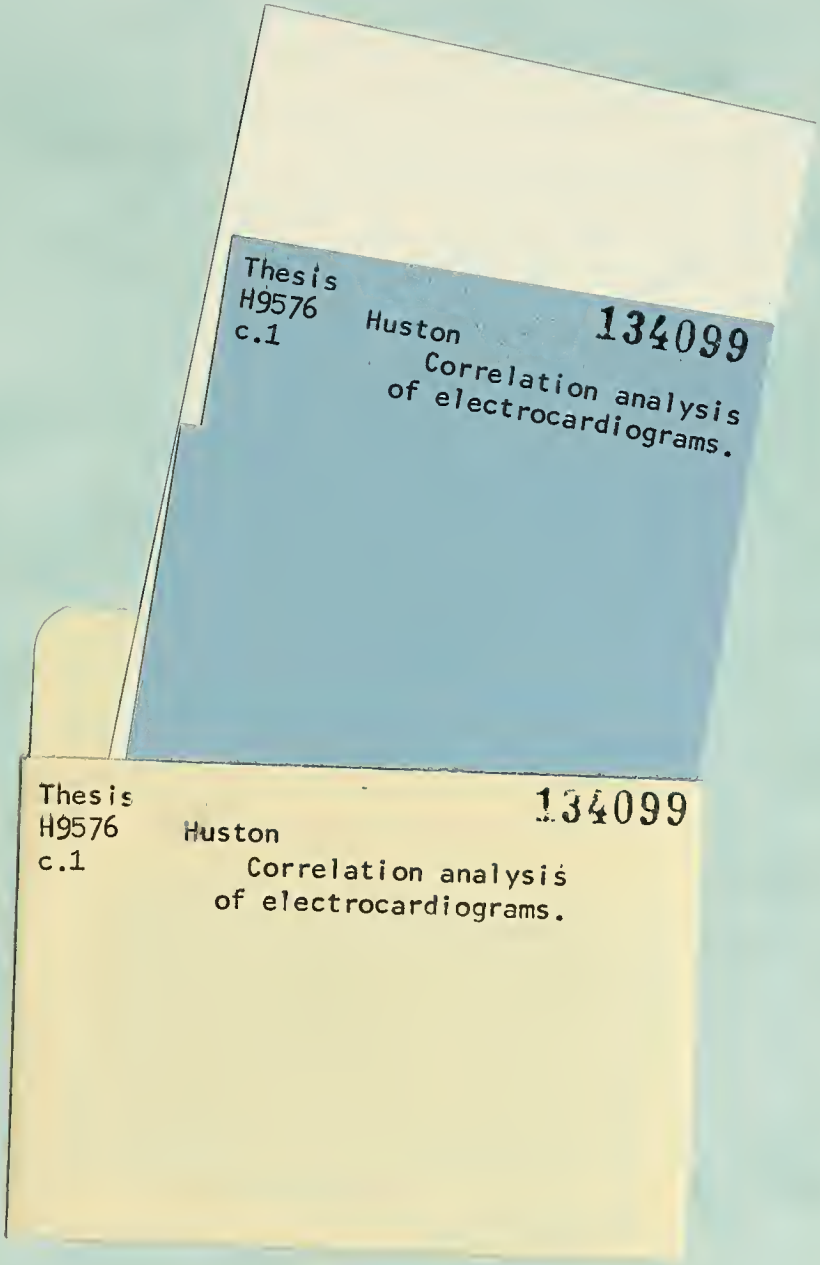
Crosscorrelate						
Electrocardiograph						
ECG, EKG						
Electrocardiographic Analysis						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